Stroke in Patients with Common Carotid Artery Dissection Secondary to Dissecting Aortic Aneurysm: an Observational Vascular Imaging Study

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Background: Common carotid artery dissection (CCAD) usually arises from the extension of an aortic arch dissection. Neurologic complications, including stroke, sometimes occur in CCAD patients. We investigated the imaging characteristics of CCAD patients with and without stroke.

Materials and Methods: From 1990 to 1999, there were eight patients diagnosed with CCAD, all due to aortic arch dissection. The clinical manifestations, findings of color-coded duplex sonography (CDS) and other vascular imaging modalities were analyzed.

Results: Ultrasonographic study of all patients revealed a visible intimal flap dissecting the common carotid artery lumen into the true and false lumina. Three patients had cerebral infarctions and one had a transient ischemic attack. Two patients had thrombi in the false lumen of the common carotid arteries on CDS. Chest radiography showed thrombi in the aortas of two patients. One patient was proved to have aortic arch dissection after a stroke.

Conclusion: Thrombi were noted in the false lumina of the common carotid arteries or aortas of all CCAD patients with stroke, but not in the patients without stroke. CDS can be used as an additional examination for the diagnosis and follow-up of patients with aortic dissection and CCAD.

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KEY WORDS: • aortic dissection • cerebral infarction • color Doppler sonography • common carotid artery dissection

INTRODUCTION

Common carotid artery dissection (CCAD) usually results from extension of a dissecting aneurysm of the aortic arch and is less commonly due to a spontaneous, traumatic or iatrogenic cause [1]. Neurologic manifestations of CCAD, including transient ischemic attack (TIA) and cerebral infarction, occur in 2.5%–7% of patients with aortic dissection [2]. Stroke after aortic dissection has important implications for initial management. Nonetheless, no characteristic finding on vascular imaging has ever

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been described in this subgroup of CCAD patients. The aim of the present observational study was to differentiate the findings of vascular imaging between patients with and without stroke.

Materials and Methods

From 1990 through 1999, there were eight patients diagnosed with CCAD (bilateral in one patient) at the neurovascular laboratory, Department of Neurology, National Taiwan University Hospital, Taipei. The medical records were reviewed in detail to study the clinical manifestations and complications. Results of vascular imaging, both radiologic and ultrasonographic findings, were specially studied for pathophysiologic correlations.

Color-coded duplex sonography (CDS) was performed in each patient using either the Diasonic VST Master Series (Milpitas, CA, USA), with a 10-MHz real-time B-mode imaging transducer and a 6-MHz pulsed Doppler transducer, or the Aloka SSD-3000 Series (Tokyo, Japan), with a 7.5-MHz real-time B-mode imaging transducer and a 5-MHz pulsed Doppler transducer. The longitudinal and transverse views of the arterial walls and lumen, intimal flaps, and intraluminal material of the cervical arteries, including the common carotid artery (CCA), internal carotid artery (ICA), external carotid artery and vertebral artery, were obtained bilaterally. The existence of intimal flaps was carefully reviewed to determine the extent of dissection, as were the Doppler signals of the true and false lumina of the CCA and ICA.

Computed tomography (CT) of the neck, chest and abdomen were performed for all patients. The extent of aortic dissection, and the presence of thrombi and intimal flaps were carefully studied. Magnetic resonance imaging (MRI) was performed in two patients. MRI was obtained on a 1.5 T system (GE Signa, Advantage, Milwaukee, WI). Complications of the cerebrovascular system were studied and categorized into cerebral infarct or TIA, using the criteria described in our previous report on the stroke registry [3, 4].

Results

Eight patients were included in this study; six men and two women, aged 41 to 83 years. They were categorized into two groups: symptomatic and asymptomatic, according to the presence or absence of cerebrovascular complications. Three patients suffered from cerebral infarction (patients 1, 2 and 3) and one (patient 4) had a TIA. Their demographic data and clinical manifestations are summarized in Table 1. All CCADs were due to extension of dissection of the aortic arch. All suffered from DeBakey Type I or II dissection. Bilateral involvement was detected in one patient (patient 1). Involvement of the right side was more common than that of the left side (7:2). Five (patients 2, 3, 4, 7 and 8) had extension of the dissection into the ICA, resulting in 30%–70% narrowing of the ICA diameter. Flow in the true lumen of the ICA was preserved in all patients. The intimal flaps within the CCA were visible on CDS in all patients (Fig. 1). The

Table 1. Demographic data and ultrasonographic findings of patients

Patient	Age (yr)/Sex	DeBakey	Side	Stroke	Days after aortic dissection
1	49/M	Type I	R	No	–
			L	L MCA infarction (R hemiparesis)	14 days
2	55/M	Type I	L	L MCA infarction	Before diagnosis of AD
3	41/M	Type I	R	R MCA infarction (L hemiparesis)	1 year
4	81/M	Type I	R	Amaurosis fugax	6 years
5	54/M	Type II	R	No	–
6	66/M	Type I	R	No	–
7	81/F	Type II	R	No	–
8	83/F	Type I	R	No	–

AD = aortic dissection; R = right; L = left; MCA = middle cerebral artery.

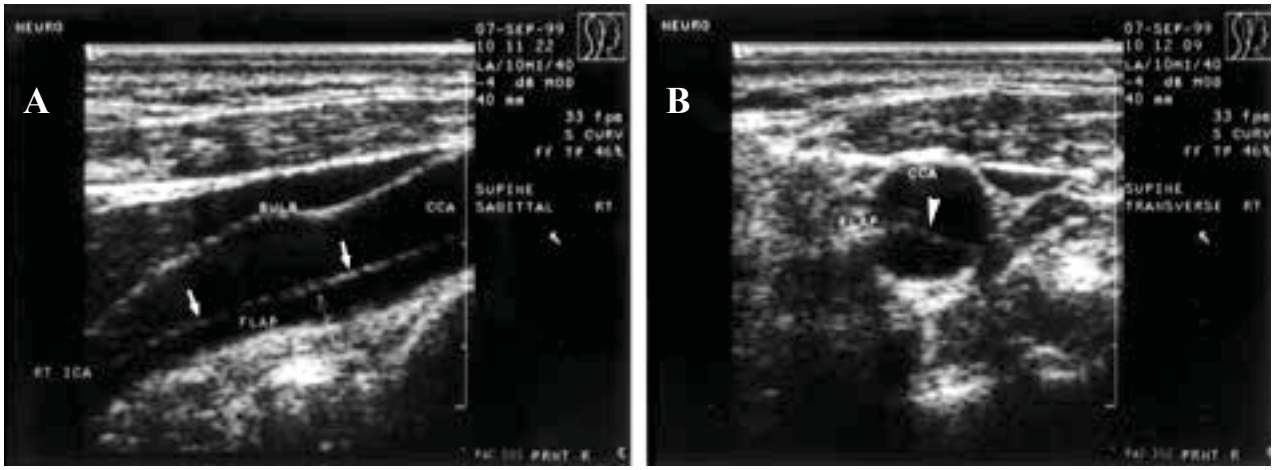

Fig. 1. *B-mode imaging of the right common carotid artery (CCA) in patient 4 demonstrated a dissecting flap (A, arrows; B, arrowhead) extending from the CCA to the internal carotid artery (ICA) in the longitudinal (A) and transverse (B) sections.*

false lumen either was filled with a thrombus (patient 1, left side and patient 2) (Fig. 2) or was perfused. No color flow signal in the thrombosed false lumen of the CCA was noted. In other patients, different color flow signals of the false lumen were seen on real-time, color-coded imaging, in the systolic and diastolic phases (Fig. 2). This represented different flow directions in the cardiac cycle, corresponding to a bidirectional reverberating flow pattern on spectral analysis. Stenosis of the true lumen of the CCA or ICA, with reduced diastolic flow velocity on spectral analysis, was demonstrated (Fig. 3).

All three patients with ischemic stroke had ipsilateral cerebral infarctions in the corresponding area of the head on CT. In addition to the common findings of CCAD on CDS, intimal flaps overlying the intramural thrombus in the ipsilateral CCA of two patients (patients 1 and 2) with cerebral infarctions were found.

CT with contrast medium of the chest and abdomen showed the extent of aortic dissection, true and false lumina, diameter of the aorta and formation of mural thrombi in patients 2 and 3. A thrombus was shown in the false lumen of the ascending aorta in patient 2. Post-stroke chest CT of patient 3 showed a mural thrombus in the retained false lumen, as well as marked dilatation of the aorta, with a maximal diameter of the aortic arch of 50 mm. These two

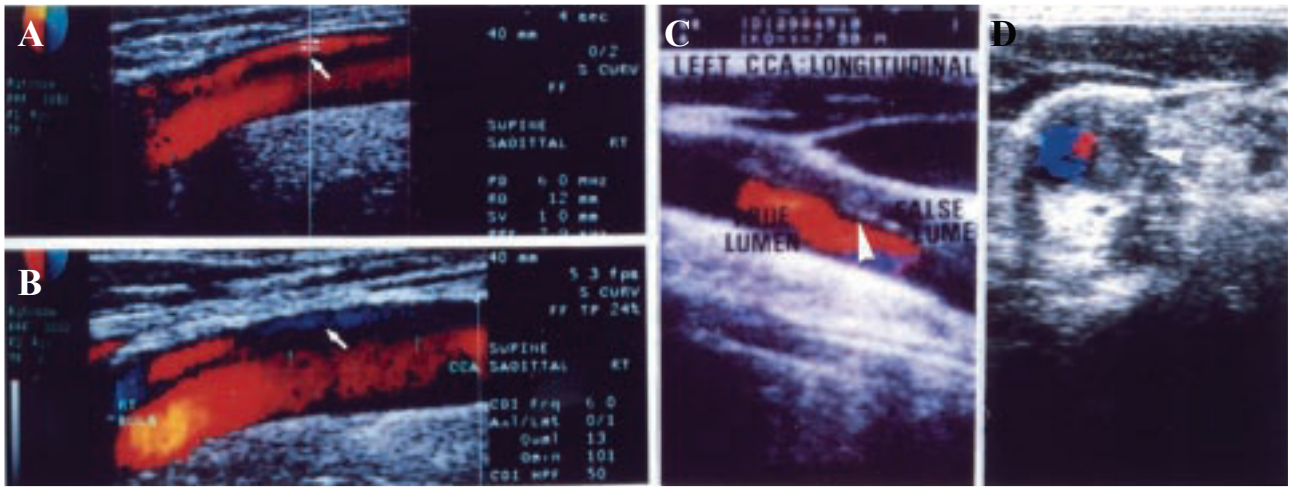

Fig. 2. *The false lumen could be perfused (A, B) or thrombosed (C, D). Different color flow signals (arrows) are shown in the false lumen in the systolic (A) and diastolic (B) phases (patient 5), representing a to-and-fro flow pattern. A thrombus in the false lumen (arrowheads) (patient 1) could be seen in both longitudinal (C) and transverse (D) views.*

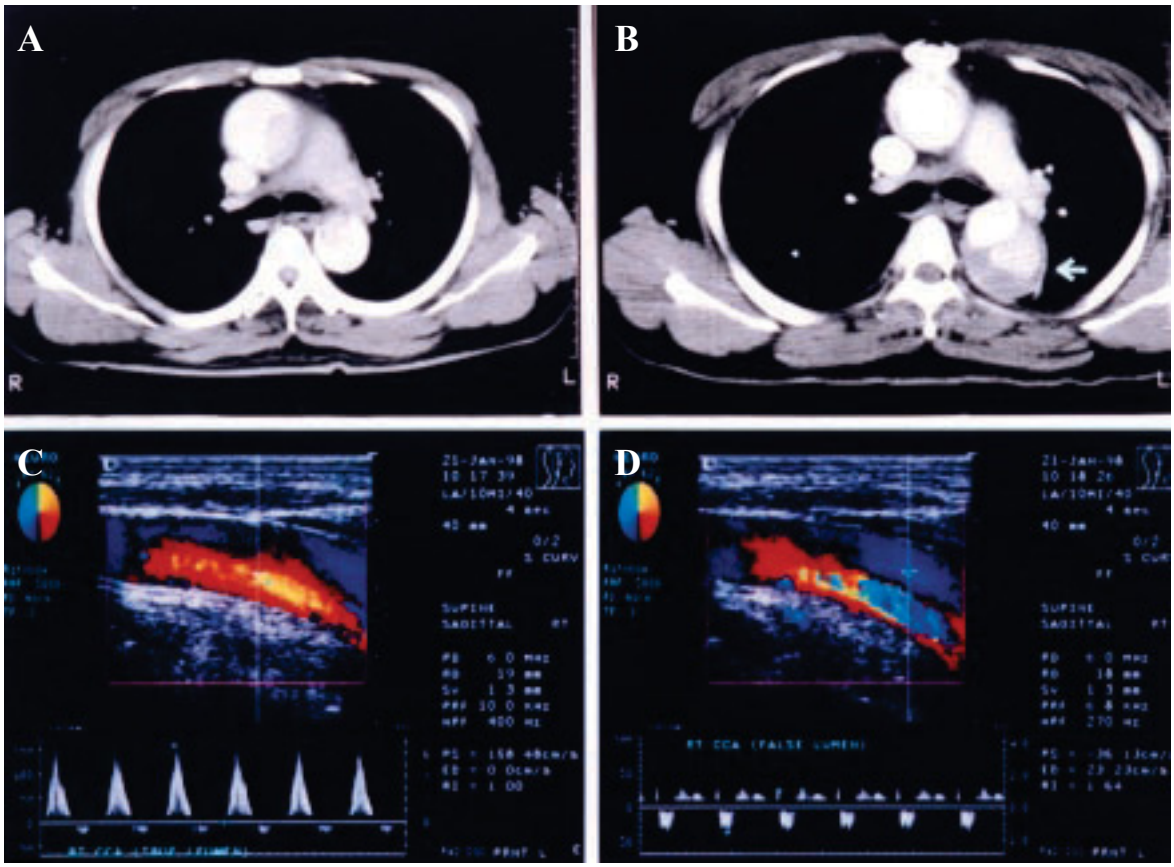

Fig. 3. *Computed tomography of the chest of patient 3 at the onset of aortic dissection (A) and after a cerebral infarction (B). A prominently dilated aortic arch with a mural thrombus in the false lumen of the residual dissection is shown (B, arrow). Spectral analysis showed different flow dynamics for the true (C) and false (D) lumina, with a high resistant flow pattern in the true lumen, and a to-and-fro flow pattern in the false lumen.*

findings were quite different from the findings on CT performed 1 year earlier (Fig. 3). MRI of patient 2 showed a thrombus in the false lumen of the ascending aorta and a recent thrombus in the left CCA. MRI of patient 8 showed a dissecting aneurysm of the ascending aorta, which extended to the right CCA.

Discussion

CCAD is being detected with increasing frequency. It was not commonly reported earlier, possibly because many of these patients are free of cerebrovascular symptoms; conventional aortography is not sensitive enough to detect CCAD, and few patients underwent CDS studies. According to previous reports, the most common cause of CCAD is extension of an aortic dissection [5].We report the same findings in our study. Among patients with aortic dissection, 15%–41% had involvement of the CCA [6–8]. Aortic angiography remains the most definitive method for confirmation of the diagnosis of aortic dissection [9], as well as dissection of the carotid and vertebral arteries [10]. Besides contrast angiography, noninvasive imaging has been widely applied to aortic dissection and carotid artery disease [9]. Duplex scanning allows the imaging of both lumina of the dissection in longitudinal and cross-sectional planes, and of the arterial wall to detect intramural thrombi. In addition to changes in anatomy, CDS provides detailed and real-time hemodynamic information. Different flow dynamics within both lumina separated by the intimal flap, as well as reentry of the false lumen, can be displayed simultaneously, thus providing an immediate diagnosis [5]. CDS has been extensively applied to cerebrovascular disease in daily practice, and can be helpful in patients with aortic dissection and CCAD.

Of CCAD patients, 15%–46% may have exceedingly variable neurologic symptoms ranging from

Table 2. Differences in sonographic and radiologic findings between symptomatic and asymptomatic cerebrovascular patients

Patient	Side	CDS study			Head CT	Chest CT		MRI
		Intimal flap	High resistant flow in true lumen	Thrombus in false lumen		Double lumina	Intraluminal thrombus	
1	R	+	+	+	L deep frontal lobe hypodensity	+	–	ND
	L	+	+	–	–			
2	L	+	+	+	L frontal lobe hypodensity	+	+ (in ascending aorta)	Thrombus in false lumen of ascending aorta and left CCA
3	R	+	+	–	R basal ganglion hypodensity	+	+ (in false lumen of the aortic arch)	ND
4	R	+	+	–	ND	+	–	ND
5	R	+	+	–	ND	+	–	ND
6	R	+	+	–	ND	+	–	ND
7	R	+	+	–	ND	+	–	ND
8	R	+	+	–	ND	+	–	Fusiform and dissecting aneurysm of ascending aorta and CCA

L = left; R = right; CDS = color-coded duplex sonography; CT = computed tomography; MRI = magnetic resonance imaging; ND = not done; CCA = common carotid artery.

dizziness to coma, due to altered intracranial hemodynamics [2]. Acute stroke was noted in 3%–7% of patients with aortic dissection [2]. Neurologic sequelae occurred in about one-third of the patients with DeBakey Types I and II dissection, and less frequently in those with DeBakey Type III dissection [2, 11, 12]. The right carotid artery was more frequently involved than the left, and this often occurred in conjunction with innominate artery dissection [2]. Carotid involvement in one autopsy series was 8%–15% [13]. Cerebral infarction could be due to common carotid occlusion or artery-to-artery embolism from a thrombus on the intimal surface of the dissected artery [14]. The CCA can be occluded at its origin by progression of the false lumen with subsequent thrombosis, or by intimal detachment at the branch orifice [14]. Cambria et al found a strong correlation between carotid occlusion and stroke in the acute phase (within 2 weeks) of aortic dissection, with 22 of 26 carotid occlusions resulting in a completed stroke [11]. They proposed that stroke and TIA rarely occur in the absence of carotid occlusion, and several case reports support this correlation [15, 16]. However, we did not observe this correlation in our study. Three patients with cerebral infarction were proved to be without CCA occlusion. On the other hand, evidence of thrombus formation was seen in all patients with infarction, either in the carotid arteries on CDS examination (patients 1 and 2), or in the aorta, as demonstrated on chest CT and MRI (patients 2 and 3). Roudaut et al monitored 32 patients with repaired DeBakey Type I and II dissections with transesophageal echocardiography and CT [17]. They found that persistent false channels may be free of thrombosis in the early period, but over time, the previously patent false channels tended to thrombose. Nakajima et al proposed that coagulopathy worsened in proportion to the degree of dilatation of the dissected aorta, especially in patients with an enlarged aorta greater than 45 mm in maximal diameter [18]. Infarction was due to artery-to-artery embolization

in two patients who had stroke 14 days and even 1 year after diagnosis of aortic dissection. Artery-to-artery embolization and coagulopathy might play an important role in the late onset of cerebral infarction after aortic dissection with CCAD. Further prospective study is necessary to confirm this viewpoint.

Among the noninvasive diagnostic modalities, CDS has several advantages. Its widespread application makes most cardiologists and neurologists familiar with it. CCAD can be immediately recognized with CDS. It can be easily used in critically ill patients in the intensive care unit, in whom MRI may be difficult to perform, and whenever contrast medium is contraindicated. Furthermore, aortic dissection with CCAD is rarely revealed from the symptoms of cerebral infarction, without the typical tearing pain [14, 19], such as in patient 2 of our study. This cause of stroke may be underestimated, and can easily be detected by CDS.

In conclusion, CCAD is not uncommon in aortic dissection, and can be overlooked in clinical practice. Since the majority of patients are neurologically asymptomatic, and aortography may miss CCAD, CDS affords an easy, safe and accurate method for detecting CCAD. Stroke resulting from CCAD has been reported to correlate with carotid artery occlusion in the acute stage. However, based on our observation, thrombosis in the residual dissection of the aorta, and subsequent artery-to-artery embolization, may play a more important role in the pathogenesis of cerebral infarction in the subacute or chronic stage. We suggest that CDS and other noninvasive radiologic studies of the aorta should be performed in the follow-up of patients with aortic dissection, not only for signs in other parts of the aorta, but also in the carotid arteries, once any symptom suggestive of cerebral ischemia is noted.

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